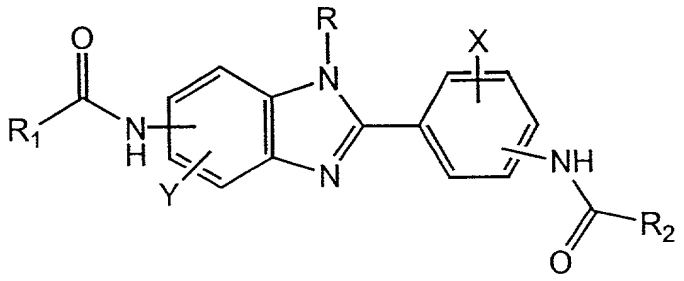
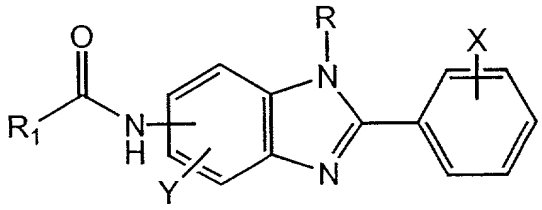
[0021] In accordance with another aspect of the present invention, there is disclosed a method of treating a mammal having a condition associated with an excess IgE level. The method comprises administering to the mammal an amount of a compound sufficient to reduced IgE levels in the mammal. The compound has the formula:

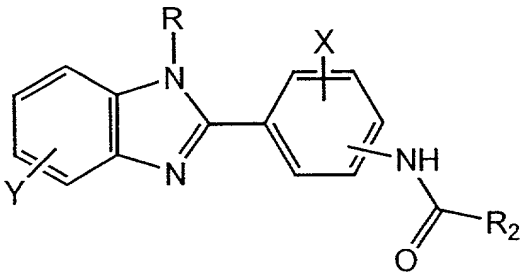
Genus A,



Genus B, and



Genus C,



[0022] wherein X and Y are independently selected from the group consisting of H, alkyl, alkoxy, aryl, substituted aryl, hydroxy, halogen, amino, alkylamino, nitro, cyano, CF_3, OCF_3. $CONH_2$, CONHR and $NHCOR_1$;

[0023] wherein R is selected from the group consisting of H, CH_3, C_2H_5, C_3H_7, C_4H_9, CH_2Ph, $CH_2C_6H_4$-F(p-), $COCH_3$, $CO_2CH_2CH_3$, aminoalkyl and dialkylaminoalkyl; and

[0024] wherein R_1 and R_2 are independently selected from the group consisting of H, aryl, heteroaryl, thiophene, pyridyl, thiazolyl, isoxazolyl, oxazolyl, pyrimidinyl, substituted aryl, substituted heteroaryl, substituted thiophene, substituted pyridyl, substituted thiazolyl, substituted isoxazolyl, substituted oxazolyl, cycloaryl, cycloheteroaryl, quinolinyl, isoquinolinyl, substituted cycloaryl, substituted cycloheteroaryl, substituted quinolinyl, substituted isoqunolinyl, multi-ring cycloaryl, multi-ring cycloheteroaryl, benzyl, heteroaryl-methyl, substituted benzyl, substituted heteroaryl-methyl alkyl, dialkylaminoalkyl, cycloalkyl, cycloalkyl containing 1-3 heteroatoms, substituted cycloalkyl, substitute cycloalkyl containing 1-3 heteroatoms, multi-ring cycloalkyl, multiring cycloalkyl containing 1-3 heteroatoms, fused-ring aliphatic, fused-ring aliphatic containing 1-3 heteroatoms, cyclopropyl, substituted cyclopropyl, cyclobutyl, substituted cyclobutyl, cyclopentyl, pyrrole, piperidine, substituted cyclopentyl, cyclohexyl, substituted cyclohexyl, cycloheptyl, substituted cycloheptyl, bicycloheptyl, substituted pyrrole, substituted piperidine, bicyclooctyl, bicyclononyl, substituted bicycloalkenyl, adamantyl, substituted adamantyl and the like, wherein at least one of R_1 and R_2 are aromatic groups or heteroaromatic groups.

[0025] The substituents on said substituted aryl, substituted heteroaryl, substituted thiophene, substituted pyridyl, substituted thiazolyl, substituted isoxazolyl, substituted oxazolyl, substituted cycloaryl, substituted cycloheteroaryl, substituted quinolinyl, substituted isoqunolinyl, substituted benzyl, substituted heteroaryl-methyl alkyl, substituted cycloalkyl, substitute cycloalkyl containing 1-3 heteroatoms, substituted cyclopropyl, cyclobutyl, substituted cyclobutyl, substituted cyclopentyl, substituted cyclohexyl, cycloheptyl, substituted cycloheptyl, bicycloheptyl, substituted pyrrole, substituted piperidine, bicyclooctyl, bicyclononyl, substituted bicycloalkenyl, adamantyl, and substituted adamantyl are independently selected from the group consisting of alkyl, aryl, CF_3, CH_3, OCH_3, OH, CN, $CONH_2$, CONHR, CONR1R2, COOR and COOH.

[0026] In a variation of the above-disclosed method, at least one additional active ingredient may be administered in conjunction with the administration of the compound. The additional active ingredient may be combined with said compound in a pharmaceutically acceptable diluent and co-administered to the mammal. The additional active ingredient may

be a short-acting β_2-adrenergic agonist selected from the group consisting of terbutaline and albuterol. In a variation, the additional active ingredient may be a long-acting β_2-adrenergic agonist selected from the group consisting of salmeterol and formoterol or an antihistamine selected from the group consisting of loratadine, azelastine and ketotifen. In another variation, the additional active ingredient may be a phosphodiesterase inhibitor, an anticholinergic agent, a corticosteroid, an inflammatory mediator release inhibitor or a leukotriene receptor antagonist.

[0027] The compound is preferably administered at a dose of about 0.01 mg to about 100 mg per kg body weight per day in divided doses of said compound for at least two consecutive days at regular periodic intervals.

[0028] Other variations within the scope of the present invention may be more fully understood with reference to the following detailed description.

Detailed Description of the Preferred Embodiment

[0029] The present invention is directed to small molecule inhibitors of IgE (synthesis and/or release) which are useful in the treatment of allergy and/or asthma or any diseases where IgE is pathogenic. The particular compounds disclosed herein were identified by their ability to suppress IgE levels in both *ex vivo* and *in vivo* assays. Development and optimization of clinical treatment regimens can be monitored by those of skill in the art by reference to the *ex vivo* and *in vivo* assays described below.

Ex Vivo Assay

[0030] This assay begins with *in vivo* antigen priming and measures secondary antibody responses *in vitro*. The basic protocol was documented and optimized for a range of parameters including: antigen dose for priming and time span following priming, number of cells cultured *in vitro*, antigen concentrations for eliciting secondary IgE (and other Ig's) response *in vitro*, fetal bovine serum (FBS) batch that will permit optimal IgE response *in vitro*, the importance of primed CD4+ T cells and hapten-specific B cells, and specificity of the ELISA assay for IgE (Marcelletti and Katz, *Cellular Immunology* 135:471-489 (1991); incorporated herein by reference).

[0031] The actual protocol utilized for this project was adapted for a more high throughput analyses. BALB/cByj mice were immunized i.p. with 10 µg DNP-KLH adsorbed